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NEWS 21 APR 28 EMBASE Controlled Term thesaurus enhanced
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=> E "TETRAHYDROCANNABINOL"/CN 25
E1      1  TETRAHYDROCANNABIDIOL/CN
E2      1  TETRAHYDROCANNABIDIOLIC ACID/CN
E3      1  --> TETRAHYDROCANNABINOL/CN
E4      1  TETRAHYDROCANNABINOL 6A(7)-HYDROXYLASE/CN
E5      1  TETRAHYDROCANNABINOLCARBOXYLIC ACID/CN
E6      2  TETRAHYDROCANNABINOLIC ACID/CN
E7      1  TETRAHYDROCANNABINOLIC ACID SYNTHASE (CANNABIS SATIVA STRAIN
001)/CN
E8      1  TETRAHYDROCANNABINOLIC ACID SYNTHASE (CANNABIS SATIVA STRAIN
005)/CN
E9      1  TETRAHYDROCANNABINOLIC ACID SYNTHASE (CANNABIS SATIVA STRAIN
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CLONE 045) /CN
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L1 1 TETRAHYDROCANNABINOL/CN

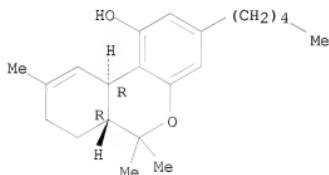
=> S L1 EXA SAM
SAMPLE IS IGNORED AS A SCOPE FOR THIS SEARCH
L2 1 TETRAHYDROCANNABINOL/CN

=> DIS L2 1

L2 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2008 ACS on STN
RN 1972-08-3 REGISTRY
ED Entered STN: 16 Nov 1984
CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-
, (6aR,10aR)- (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-
, (6aR-trans)-
CN Cannabinol, Δ 1-tetrahydro- (7CI)
CN Cannabinol, tetrahydro- (6CI)
OTHER NAMES:
CN (-)- Δ 1-Tetrahydrocannabinol
CN (-)- Δ 9-Tetrahydrocannabinol
CN (-)- Δ 9-trans-Tetrahydrocannabinol
CN (-)-3,4-trans- Δ 1-Tetrahydrocannabinol
CN (-)-trans- Δ 1-Tetrahydrocannabinol
CN (-)-trans- Δ 9-Tetrahydrocannabinol
CN (-)-trans- Δ 9-THC
CN (1)- Δ 1-Tetrahydrocannabinol
CN Δ 1-Tetrahydrocannabinol
CN Δ 1-THC
CN Δ 9-Tetrahydrocannabinol
CN Δ 9-THC
CN Δ 9-trans-Tetrahydrocannabinol
CN Abbott 40566
CN Dronabinol
CN 1- Δ 1-trans-Tetrahydrocannabinol
CN 1-trans- Δ 9-Tetrahydrocannabinol
CN Marinol
CN NSC 134454
CN QCD 84924
CN SP 104
CN Tetrahydrocannabinol
CN THC

CN trans-(-)- Δ^9 -Tetrahydrocannabinol
 CN trans- Δ^9 -Tetrahydrocannabinol
 FS STEREOSEARCH
 DR 14146-29-3, 14146-43-1, 1363-19-5, 5957-27-7, 26108-45-2
 MF C21 H30 O2
 CI COM
 LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*,
 BIOSIS, BIOTECHNO, CA, CABAB, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS,
 CHEMLIST, CIN, CSChem, DDFU, DRUGU, EMBASE, HSDB*, IFICDB, IFIPAT,
 IFIUDB, IMSCOSEARCH, IMSDRUGNEWS, IMSPATENTS, IMSPRODUCT, IMSRESEARCH,
 IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, PHAR, PIRA, PROMT, PROUSDDR,
 PS, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL,
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 (*File contains numerically searchable property data)
 Other Sources: WHO

Absolute stereochemistry. Rotation (-).



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 140 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
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 4 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> file medline caplus wpids uspatfull	SINCE FILE	TOTAL
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=> s 12
 L3 9938 L2

=> s 13 and glioblastoma
 L4 23 L3 AND GLIOBLASTOMA

=> d 14 1-23 ibib, abs, hitstr

L4 ANSWER 1 OF 23 MEDLINE on STN
ACCESSION NUMBER: 2008180791 MEDLINE
DOCUMENT NUMBER: PubMed ID: 18339876
TITLE: Cannabinoids inhibit glioma cell invasion by
down-regulating matrix metalloproteinase-2 expression.
AUTHOR: Blazquez Cristina; Salazar Maria; Carracedo Arkaitz;
Lorente Mar; Egia Ainara; Gonzalez-Feria Luis; Haro Amador;
Velasco Guillermo; Guzman Manuel
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology I, School
of Biology, Complutense University, Madrid, Spain.
SOURCE: Cancer research, (2008 Mar 15) Vol. 68, No. 6, pp. 1945-52.
Journal code: 2984705R. E-ISSN: 1538-7445.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200804
ENTRY DATE: Entered STN: 15 Mar 2008
Last Updated on STN: 10 Apr 2008
Entered Medline: 9 Apr 2008

AB Cannabinoids, the active components of *Cannabis sativa* L. and their derivatives, inhibit tumor growth in laboratory animals by inducing apoptosis of tumor cells and impairing tumor angiogenesis. It has also been reported that these compounds inhibit tumor cell spreading, but the molecular targets of this cannabinoid action remain elusive. Here, we evaluated the effect of cannabinoids on matrix metalloproteinase (MMP) expression and its effect on tumor cell invasion. Local administration of Delta(9)-tetrahydrocannabinol (THC), the major active ingredient of cannabis, down-regulated MMP-2 expression in gliomas generated in mice, as determined by Western blot, immunofluorescence, and real-time quantitative PCR analyses. This cannabinoid-induced inhibition of MMP-2 expression in gliomas (a) was MMP-2-selective, as levels of other MMP family members were unaffected; (b) was mimicked by JWH-133, a CB(2) cannabinoid receptor-selective agonist that is devoid of psychoactive side effects; (c) was abrogated by fumonisin B1, a selective inhibitor of ceramide biosynthesis; and (d) was also evident in two patients with recurrent glioblastoma multiforme. THC inhibited MMP-2 expression and cell invasion in cultured glioma cells. Manipulation of MMP-2 expression by RNA interference and cDNA overexpression experiments proved that down-regulation of this MMP plays a critical role in THC-mediated inhibition of cell invasion. Cannabinoid-induced inhibition of MMP-2 expression and cell invasion was prevented by blocking ceramide biosynthesis and by knocking-down the expression of the stress protein p8. As MMP-2 up-regulation is associated with high progression and poor prognosis of gliomas and many other tumors, MMP-2 down-regulation constitutes a new hallmark of cannabinoid antitumoral activity.

L4 ANSWER 2 OF 23 MEDLINE on STN
ACCESSION NUMBER: 2008000154 MEDLINE
DOCUMENT NUMBER: PubMed ID: 17675107
TITLE: Down-regulation of tissue inhibitor of metalloproteinases-1
in gliomas: a new marker of cannabinoid antitumoral
activity?
AUTHOR: Blazquez Cristina; Carracedo Arkaitz; Salazar Maria;
Lorente Mar; Egia Ainara; Gonzalez-Feria Luis; Haro Amador;
Velasco Guillermo; Guzman Manuel
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology I, School
of Biology, Complutense University, 28040 Madrid, Spain.

SOURCE: Neuropharmacology, (2008 Jan) Vol. 54, No. 1, pp. 235-43.
Electronic Publication: 2007-07-01.
Journal code: 0236217. ISSN: 0028-3908.

PUB. COUNTRY: England: United Kingdom
(CLINICAL TRIAL)

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200804

ENTRY DATE: Entered STN: 1 Jan 2008
Last Updated on STN: 19 Apr 2008
Entered Medline: 18 Apr 2008

AB Cannabinoids, the active components of Cannabis sativa L. and their derivatives, inhibit tumor growth in laboratory animals by inducing apoptosis of tumor cells and inhibiting tumor angiogenesis. It has also been reported that cannabinoids inhibit tumor cell invasiveness, but the molecular targets of this cannabinoid action remain elusive. Here we evaluated the effects of cannabinoids on the expression of tissue inhibitors of metalloproteinases (TIMPs), which play critical roles in the acquisition of migrating and invasive capacities by tumor cells. Local administration of Delta(9)-tetrahydrocannabinol (THC), the major active ingredient of cannabis, down-regulated TIMP-1 expression in mice bearing subcutaneous gliomas, as determined by Western blot and immunofluorescence analyses. This cannabinoid-induced inhibition of TIMP-1 expression in gliomas (i) was mimicked by JWH-133, a selective CB(2) cannabinoid receptor agonist that is devoid of psychoactive side effects, (ii) was abrogated by fumonisin B1, a selective inhibitor of ceramide synthesis de novo, and (iii) was also evident in two patients with recurrent glioblastoma multiforme (grade IV astrocytoma). THC also depressed TIMP-1 expression in cultures of various human glioma cell lines as well as in primary tumor cells obtained from a glioblastoma multiforme patient. This action was prevented by pharmacological blockade of ceramide biosynthesis and by knocking-down the expression of the stress protein p8. As TIMP-1 up-regulation is associated with high malignancy and negative prognosis of numerous cancers, TIMP-1 down-regulation may be a hallmark of cannabinoid-induced inhibition of glioma progression.

L4 ANSWER 3 OF 23 MEDLINE on STN
ACCESSION NUMBER: 2006412347 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16804518

TITLE: A pilot clinical study of Delta9-tetrahydrocannabinol in patients with recurrent glioblastoma multiforme.

AUTHOR: Guzman M; Duarte M J; Blazquez C; Ravina J; Rosa M C; Galve-Roperh I; Sanchez C; Velasco G; Gonzalez-Feria L

CORPORATE SOURCE: Department of Biochemistry and Molecular Biology I, School of Biology, Complutense University, Madrid 28040, Spain..
mgp@bbm1.ucm.es

SOURCE: British journal of cancer, (2006 Jul 17) Vol. 95, No. 2, pp. 197-203. Electronic Publication: 2006-06-27.
Journal code: 0370635. ISSN: 0007-0920.

PUB. COUNTRY: England: United Kingdom
(CLINICAL TRIAL)

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200611

ENTRY DATE: Entered STN: 13 Jul 2006
Last Updated on STN: 7 Nov 2006
Entered Medline: 6 Nov 2006

AB Delta(9)-Tetrahydrocannabinol (THC) and other cannabinoids inhibit tumour growth and angiogenesis in animal models, so their potential application as antitumoral drugs has been suggested. However, the antitumoral effect of cannabinoids has never been tested in humans. Here we report the first clinical study aimed at assessing cannabinoid antitumoral action, specifically a pilot phase I trial in which nine patients with recurrent glioblastoma multiforme were administered THC intratumorally. The patients had previously failed standard therapy (surgery and radiotherapy) and had clear evidence of tumour progression. The primary end point of the study was to determine the safety of intracranial THC administration. We also evaluated THC action on the length of survival and various tumour-cell parameters. A dose escalation regimen for THC administration was assessed. Cannabinoid delivery was safe and could be achieved without overt psychoactive effects. Median survival of the cohort from the beginning of cannabinoid administration was 24 weeks (95% confidence interval: 15-33). Delta(9)-Tetrahydrocannabinol inhibited tumour-cell proliferation in vitro and decreased tumour-cell Ki67 immunostaining when administered to two patients. The fair safety profile of THC, together with its possible antiproliferative action on tumour cells reported here and in other studies, may set the basis for future trials aimed at evaluating the potential antitumoral activity of cannabinoids.

L4 ANSWER 4 OF 23 MEDLINE on STN
ACCESSION NUMBER: 2006209507 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16616335
TITLE: The stress-regulated protein p8 mediates cannabinoid-induced apoptosis of tumor cells.
AUTHOR: Carracedo Arkaitz; Lorente Mar; Egia Ainara; Blazquez Cristina; Garcia Stephane; Giroux Valentin; Malicet Cedric; Villuendas Raquel; Gironella Meritxell; Gonzalez-Feria Luis; Piris Miguel Angel; Iovanna Juan L; Guzman Manuel; Velasco Guillermo
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology I, School of Biology, Complutense University, 28040 Madrid, Spain.
SOURCE: Cancer cell, (2006 Apr) Vol. 9, No. 4, pp. 301-12.
Journal code: 101130617. ISSN: 1535-6108.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200605
ENTRY DATE: Entered STN: 18 Apr 2006
Last Updated on STN: 17 May 2006
Entered Medline: 16 May 2006

AB One of the most exciting areas of current research in the cannabinoid field is the study of the potential application of these compounds as antitumoral drugs. Here, we describe the signaling pathway that mediates cannabinoid-induced apoptosis of tumor cells. By using a wide array of experimental approaches, we identify the stress-regulated protein p8 (also designated as candidate of metastasis 1) as an essential mediator of cannabinoid antitumoral action and show that p8 upregulation is dependent on de novo-synthesized ceramide. We also observe that p8 mediates its apoptotic effect via upregulation of the endoplasmic reticulum stress-related genes ATF-4, CHOP, and TRB3. Activation of this pathway may constitute a potential therapeutic strategy for inhibiting tumor growth.

L4 ANSWER 5 OF 23 MEDLINE on STN
ACCESSION NUMBER: 2005413320 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16078104
TITLE: Cannabinoids selectively inhibit proliferation and induce

AUTHOR: death of cultured human glioblastoma multiforme cells.
McAllister Sean D; Chan Calvin; Taft Ryan J; Luu Tri; Abood Mary E; Moore Dan H; Aldape Ken; Yount Garret
CORPORATE SOURCE: California Pacific Medical Center Research Institute, 475 Brannan St., Suite 220, San Francisco, CA 94107, USA.. mcallis@sutterhealth.org
CONTRACT NUMBER: 05274 (United States NCCAM)
09978
AT00643
SOURCE: Journal of neuro-oncology, (2005 Aug) Vol. 74, No. 1, pp. 31-40.
Journal code: 8309335. ISSN: 0167-594X.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200510
ENTRY DATE: Entered STN: 4 Aug 2005
Last Updated on STN: 15 Oct 2005
Entered Medline: 14 Oct 2005

AB Normal tissue toxicity limits the efficacy of current treatment modalities for glioblastoma multiforme (GBM). We evaluated the influence of cannabinoids on cell proliferation, death, and morphology of human GBM cell lines and in primary human glial cultures, the normal cells from which GBM tumors arise. The influence of a plant derived cannabinoid agonist, Delta(9)-tetrahydrocannabinol Delta(9)-THC, and a potent synthetic cannabinoid agonist, WIN 55,212-2, were compared using time lapse microscopy. We discovered that Delta(9)-THC decreases cell proliferation and increases cell death of human GBM cells more rapidly than WIN 55,212-2. Delta(9)-THC was also more potent at inhibiting the proliferation of GBM cells compared to WIN 55,212-2. The effects of Delta(9)-THC and WIN 55,212-2 on the GBM cells were partially the result of cannabinoid receptor activation. The same concentration of Delta(9)-THC that significantly inhibits proliferation and increases death of human GBM cells has no significant impact on human primary glial cultures. Evidence of selective efficacy with WIN 55,212-2 was also observed but the selectivity was less profound, and the synthetic agonist produced a greater disruption of normal cell morphology compared to Delta(9)-THC.

L4 ANSWER 6 OF 23 MEDLINE on STN
ACCESSION NUMBER: 2004133485 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15026328
TITLE: Cannabinoids induce cancer cell proliferation via tumor necrosis factor alpha-converting enzyme (TACE/ADAM17)-mediated transactivation of the epidermal growth factor receptor.
AUTHOR: Hart Stefan; Fischer Oliver M; Ullrich Axel
CORPORATE SOURCE: Department of Molecular Biology, Max-Planck-Institute of Biochemistry, Am Klopferspitz 18A, D-82152 Martinsried, Germany.
SOURCE: Cancer research, (2004 Mar 15) Vol. 64, No. 6, pp. 1943-50.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 200404
ENTRY DATE: Entered STN: 18 Mar 2004
Last Updated on STN: 9 Apr 2004
Entered Medline: 8 Apr 2004

AB Cannabinoids, the active components of marijuana and their endogenous counterparts were reported as useful analgesic agents to accompany primary cancer treatment by preventing nausea, vomiting, and pain and by stimulating appetite. Moreover, they have been shown to inhibit cell growth and to induce apoptosis in tumor cells. Here, we demonstrate that anandamide, Delta(9)-tetrahydrocannabinol (THC), HU-210, and Win55,212-2 promote mitogenic kinase signaling in cancer cells. Treatment of the glioblastoma cell line U373-MG and the lung carcinoma cell line NCI-H292 with nanomolar concentrations of THC led to accelerated cell proliferation that was completely dependent on metalloprotease and epidermal growth factor receptor (EGFR) activity. EGFR signal transactivation was identified as the mechanistic link between cannabinoid receptors and the activation of the mitogen-activated protein kinases extracellular signal-regulated kinase 1/2 as well as prosurvival protein kinase B (Akt/PKB) signaling. Depending on the cellular context, signal cross-communication was mediated by shedding of proAmphiregulin (proAR) and/or proHeparin-binding epidermal growth factor-like growth factor (proHB-EGF) by tumor necrosis factor alpha converting enzyme (TACE/ADAM17). Taken together, our data show that concentrations of THC comparable with those detected in the serum of patients after THC administration accelerate proliferation of cancer cells instead of apoptosis and thereby contribute to cancer progression in patients.

L4 ANSWER 7 OF 23 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2008:43490 CAPLUS
DOCUMENT NUMBER: 148:135980
TITLE: Blood levels of insulin-like growth factor-binding protein 2 as a marker for monitoring the effectiveness of inhibitors of insulin-like growth factor I receptors in cancer therapy
INVENTOR(S): Wang, Yan
PATENT ASSIGNEE(S): Schering Corporation, USA
SOURCE: PCT Int. Appl., 133pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2008005469	A2	20080110	WO 2007-US15423	20070629
WO 2008005469	A3	20080228		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GO, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRIORITY APPLN. INFO.: US 2006-818004P P 20060630
AB The present invention provides method for quickly and conveniently determining if a given treatment regimen of insulin-like growth factor I receptor

(IGF1R) inhibitor is sufficient, e.g., to saturate IGF1 R receptors in the body of a subject. Blood levels of insulin-like growth factor-binding protein 2 (IGFBP2) are shown to be strongly correlated with the effectiveness of IGF1R receptor therapy. Several clin. relevant detns. may be made based on this point, including, for example, whether the dosage of the regimen is sufficient or should be increased. The relationship is demonstrated using animal xenograft models of neuroblastoma. Treatment with monoclonal antibodies to IGF1R lowered the blood levels of IGFBP2. The level of IGFBP2 correlated with the tumor size.

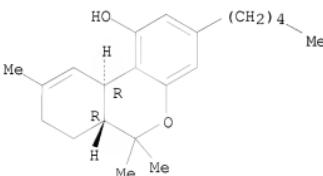
IT 1972-08-3, Dronabinol

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses) (cancer therapy using; blood levels of IGBP2 as marker for monitoring effectiveness of inhibitors of IGF1 receptors in cancer therapy)

RN 1972-08-3 CAPLUS

CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L4 ANSWER 8 OF 23 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2008:13451 CAPLUS

DOCUMENT NUMBER: 148:182733

TITLE: Down-regulation of tissue inhibitor of metalloproteinases-1 in gliomas: a new marker of cannabinoid antitumoral activity?

AUTHOR(S): Blazquez, Cristina; Carracedo, Arkaitz; Salazar, Maria; Lorente, Mar; Egia, Ainara; Gonzalez-Feria, Luis; Haro, Amador; Velasco, Guillermo; Guzman, Manuel

CORPORATE SOURCE: Department of Biochemistry and Molecular Biology I, School of Biology, Complutense University, Madrid, 28040, Spain

SOURCE: Neuropharmacology (2007), Volume Date 2008, 54(1), 235-243

PUBLISHER: CODEN: NEPHBW; ISSN: 0028-3908
Elsevier B.V.

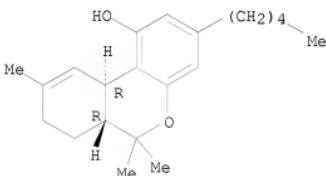
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Cannabinoids, the active components of Cannabis sativa L. and their derivs., inhibit tumor growth in laboratory animals by inducing apoptosis of tumor cells and inhibiting tumor angiogenesis. It has also been reported that cannabinoids inhibit tumor cell invasiveness, but the mol. targets of this cannabinoid action remain elusive. Here we evaluated the effects of cannabinoids on the expression of tissue inhibitors of metalloproteinases (TIMPs), which play critical roles in the acquisition of migrating and invasive capacities by tumor cells. Local administration of Δ^9 -tetrahydrocannabinol (THC), the major active ingredient of cannabis, down-regulated TIMP-1 expression in mice bearing s.c. gliomas, as determined by Western blot and immunofluorescence analyses. This

cannabinoid-induced inhibition of TIMP-1 expression in gliomas (i) was mimicked by JWH-133, a selective CB2 cannabinoid receptor agonist that is devoid of psychoactive side effects, (ii) was abrogated by fumonisin B1, a selective inhibitor of ceramide synthesis de novo, and (iii) was also evident in two patients with recurrent glioblastoma multiforme (grade IV astrocytoma). THC also depressed TIMP-1 expression in cultures of various human glioma cell lines as well as in primary tumor cells obtained from a glioblastoma multiforme patient. This action was prevented by pharmacol. blockade of ceramide biosynthesis and by knocking-down the expression of the stress protein p8. As TIMP-1 up-regulation is associated with high malignancy and neg. prognosis of numerous cancers, TIMP-1 down-regulation may be a hallmark of cannabinoid-induced inhibition of glioma progression.

IT 1972-08-3, Δ^9 -Tetrahydrocannabinol
 RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (down-regulation of tissue inhibitor of metalloproteinases-1 in gliomas as a new marker of cannabinoid antitumoral activity)
 RN 1972-08-3 CAPLUS
 CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 9 OF 23 CAPLUS COPYRIGHT 2008 ACS on STN
 ACCESSION NUMBER: 2006:692024 CAPLUS
 DOCUMENT NUMBER: 146:134714
 TITLE: A pilot clinical study of Δ^9 -tetrahydrocannabinol in patients with recurrent glioblastoma multiforme
 AUTHOR(S): Guzman, M.; Duarte, M. J.; Blazquez, C.; Ravina, J.; Rosa, M. C.; Galve-Roperh, I.; Sanchez, C.; Velasco, G.; Gonzalez-Feria, L.
 CORPORATE SOURCE: Department of Biochemistry and Molecular Biology I, School of Biology, Complutense University, Madrid, 28040, Spain
 SOURCE: British Journal of Cancer (2006), 95(2), 197-203
 CODEN: BJCAAI; ISSN: 0007-0920
 PUBLISHER: Nature Publishing Group
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Δ^9 -Tetrahydrocannabinol (THC) and other cannabinoids inhibit tumor growth and angiogenesis in animal models, so their potential application as antitumoral drugs has been suggested. However, the antitumoral effect of cannabinoids has never been tested in humans. Here we report the first clin. study aimed at assessing cannabinoid antitumoral action, specifically a pilot phase I trial in which nine patients with recurrent

glioblastoma multiforme were administered THC intratumorally. The patients had previously failed standard therapy (surgery and radiotherapy) and had clear evidence of tumor progression. The primary end point of the study was to determine the safety of intracranial THC administration. We also evaluated THC action on the length of survival and various tumor-cell parameters. A dose escalation regimen for THC administration was assessed. Cannabinoid delivery was safe and could be achieved without overt psychoactive effects. Median survival of the cohort from the beginning of cannabinoid administration was 24 wk (95% confidence interval: 15–33). $\Delta 9$ -Tetrahydrocannabinol inhibited tumor-cell proliferation in vitro and decreased tumor-cell Ki67 immunostaining when administered to two patients. The fair safety profile of THC, together with its possible antiproliferative action on tumor cells reported here and in other studies, may set the basis for future trials aimed at evaluating the potential antitumoral activity of cannabinoids.

IT 1972-08-3, $\Delta 9$ -Tetrahydrocannabinol

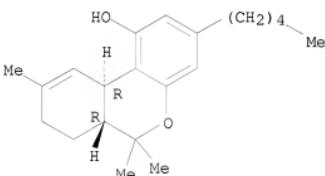
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(phase I trial of dose escalation of $\Delta 9$ -tetrahydrocannabinol delivery was safe, inhibited cell proliferation, tumor growth and vascularization, CB1 and CB2 receptor expressions in patient with recurrent glioblastoma multiforme)

RN 1972-08-3 CAPLUS

CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 10 OF 23 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2006:393185 CAPLUS

DOCUMENT NUMBER: 144:404250

TITLE: The stress-regulated protein p8 mediates cannabinoid-induced apoptosis of tumor cells
Carracedo, Arkaitz; Lorente, Mari; Egia, Ainara; Blazquez, Cristina; Garcia, Stephane; Giroux, Valentin; Malicet, Cedric; Villuendas, Raquel; Gironella, Meritxell; Gonzalez-Feria, Luis; Piris, Miguel Angel; Iovanna, Juan L.; Guzman, Manuel; Velasco, Guillermo

CORPORATE SOURCE: Department of Biochemistry and Molecular Biology I, School of Biology, Complutense University, Madrid, 28040, Spain

SOURCE: Cancer Cell (2006), 9(4), 301-312
CODEN: CCAECI; ISSN: 1535-6108

PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB One of the most exciting areas of current research in the cannabinoid field is the study of the potential application of these compds. as antitumoral drugs. Here, we describe the signaling pathway that mediates cannabinoid-induced apoptosis of tumor cells. By using a wide array of exptl. approaches, we identify the stress-regulated protein p8 (also designated as candidate of metastasis 1) as an essential mediator of cannabinoid antitumoral action and show that p8 upregulation is dependent on de novo-synthesized ceramide. We also observe that p8 mediates its apoptotic effect via upregulation of the endoplasmic reticulum stress-related genes ATF-4, CHOP, and TRB3. Activation of this pathway may constitute a potential therapeutic strategy for inhibiting tumor growth.

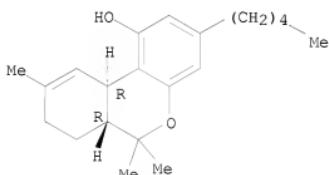
IT 1972-08-3, Δ^9 -Tetrahydrocannabinol

RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(stress-regulated protein p8 mediates cannabinoid-induced apoptosis of tumor cells)

RN 1972-08-3 CAPLUS

CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 11 OF 23 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2005:1303230 CAPLUS

DOCUMENT NUMBER: 144:304660

TITLE: Cannabinoids selectively inhibit proliferation and induce death of cultured human glioblastoma multiforme cells

AUTHOR(S): McAlister, Sean D.; Chan, Calvin; Taft, Ryan J.; Luu, Tri; Abood, Mary E.; Moore, Dan H.; Aldape, Ken; Yount, Garret

CORPORATE SOURCE: California Pacific Medical Center Research Institute, San Francisco, CA, USA

SOURCE: Journal of Neuro-Oncology (2005), 74(1), 31-40
CODEN: JNODD2; ISSN: 0167-594X

PUBLISHER: Springer

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Normal tissue toxicity limits the efficacy of current treatment modalities for glioblastoma multiforme (GBM). We evaluated the influence of cannabinoids on cell proliferation, death, and morphol. of human GBM cell lines and in primary human glial cultures, the normal cells from which GBM tumors arise. The influence of a plant derived cannabinoid agonist, Δ^9 -tetrahydrocannabinol (Δ^9 -THC), and a potent synthetic cannabinoid agonist, WIN 55,212-2, were compared using time lapse microscopy. We discovered that Δ^9 -THC decreases cell

proliferation and increases cell death of human GBM cells more rapidly than WIN 55,212-2. Δ^9 -THC was also more potent at inhibiting the proliferation of GBM cells compared to WIN 55,212-2. The effects of Δ^9 -THC and WIN 55,212-2 on the GBM cells were partially the result of cannabinoid receptor activation. The same concentration of Δ^9 -THC that significantly inhibits proliferation and increases death of human GBM cells has no significant impact on human primary glial cultures. Evidence of selective efficacy with WIN 55,212-2 was also observed but the selectivity was less profound, and the synthetic agonist produced a greater disruption of normal cell morphol. compared to Δ^9 -THC.

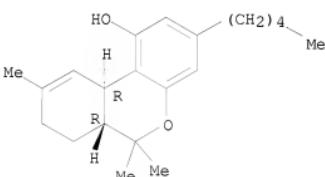
IT 1972-08-3, Δ^9 -Tetrahydrocannabinol

RL: NPO (Natural product occurrence); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); USES (Uses) (Δ^9 -THC decreased proliferation and increased death of human GBM cells rapidly than WIN 55,212-2 with involvement of cannabinoid receptors activation but WIN 55,212-2 greatly disrupted cell morphol. compared to Δ^9 -THC)

RN 1972-08-3 CAPLUS

CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 12 OF 23 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:672684 CAPLUS

DOCUMENT NUMBER: 141:218491

TITLE: Cannabinoids Inhibit the Vascular Endothelial Growth Factor Pathway in Gliomas

AUTHOR(S): Blazquez, Cristina; Gonzalez-Feria, Luis; Alvarez, Luis; Haro, Amador; Casanova, M. Llanos; Guzman, Manuel

CORPORATE SOURCE: Department of Biochemistry and Molecular Biology I, School of Biology, Complutense Univ., Madrid, Spain

SOURCE: Cancer Research (2004), 64(16), 5617-5623

CODEN: CNREA8; ISSN: 0008-5472

PUBLISHER: American Association for Cancer Research

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Cannabinoids inhibit tumor angiogenesis in mice, but the mechanism of their antiangiogenic action is still unknown. Because the vascular endothelial growth factor (VEGF) pathway plays a critical role in tumor angiogenesis, here the authors studied whether cannabinoids affect it. As a first approach, cDNA array anal. showed that cannabinoid administration to mice bearing s.c. gliomas lowered the expression of various VEGF pathway-related genes. The use of other methods (ELISA, Western blotting, and confocal microscopy) provided addnl. evidence that cannabinoids depressed the VEGF pathway by decreasing the production of VEGF and the

activation of VEGF receptor (VEGFR)-2, the most prominent VEGF receptor, in cultured glioma cells and in mouse gliomas. Cannabinoid-induced inhibition of VEGF production and VEGFR-2 activation was abrogated both in vitro and in vivo by pharmacol. blockade of ceramide biosynthesis. These changes in the VEGF pathway were paralleled by changes in tumor size. Moreover, intratumoral administration of the cannabinoid Δ^9 -tetrahydrocannabinol to two patients with glioblastoma multiforme (grade IV astrocytoma) decreased VEGF levels and VEGFR-2 activation in the tumors. Because blockade of the VEGF pathway constitutes one of the most promising antitumoral approaches currently available, the present findings provide a novel pharmacol. target for cannabinoid-based therapies.

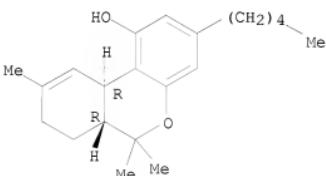
IT 1972-08-3, Δ^9 -Tetrahydrocannabinol

RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cannabinoids inhibit the vascular endothelial growth factor pathway in gliomas in relation to inhibition of tumor angiogenesis)

RN 1972-08-3 CAPLUS

CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 13 OF 23 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:214360 CAPLUS

DOCUMENT NUMBER: 140:350165

TITLE: Cannabinoids Induce Cancer Cell Proliferation via Tumor Necrosis Factor α -Converting Enzyme (TACE/ADAM17)-Mediated Transactivation of the Epidermal Growth Factor Receptor

AUTHOR(S): Hart, Stefan; Fischer, Oliver M.; Ullrich, Axel

CORPORATE SOURCE: Department of Molecular Biology, Max-Planck-Institute of Biochemistry, Martinsried, Germany

SOURCE: Cancer Research (2004), 64(6), 1943-1950

PUBLISHER: CODEN: CNREAB; ISSN: 0008-5472

DOCUMENT TYPE: American Association for Cancer Research
Journal

LANGUAGE: English

AB Cannabinoids, the active components of marijuana and their endogenous counterparts were reported as useful analgesic agents to accompany primary cancer treatment by preventing nausea, vomiting, and pain and by stimulating appetite. Moreover, they have been shown to inhibit cell growth and to induce apoptosis in tumor cells. Here, we demonstrate that anandamide, Δ^9 -tetrahydrocannabinol (THC), HU-210, and Win55212-2 promote mitogenic kinase signaling in cancer cells. Treatment of the glioblastoma cell line U373-MG and the lung carcinoma cell line NCI-H292 with nanomolar concns. of THC led to accelerated cell

proliferation that was completely dependent on metalloprotease and epidermal growth factor receptor (EGFR) activity. EGFR signal transactivation was identified as the mechanistic link between cannabinoid receptors and the activation of the mitogen-activated protein kinases extracellular signal-regulated kinase 1/2 as well as prosurvival protein kinase B (Akt/PKB) signaling. Depending on the cellular context, signal cross-communication was mediated by shedding of proAmphiregulin (pro Ar) and/or proHeparin-binding epidermal growth factor-like growth factor (proHB-EGF) by tumor necrosis factor α converting enzyme (TACE/ADAM17). Taken together, our data show that concns. of THC comparable with those detected in the serum of patients after THC administration accelerate proliferation of cancer cells instead of apoptosis and thereby contribute to cancer progression in patients.

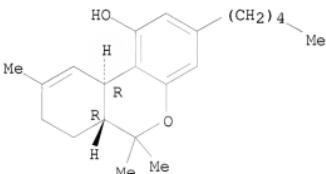
IT 1972-08-3, A9-Tetrahydrocannabinol

RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (cannabinoids induce cancer cell proliferation via tumor necrosis factor α -converting enzyme (TACE/ADAM17)-mediated transactivation of epidermal growth factor receptor)

RN 1972-08-3 CAPLUS

CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 14 OF 23 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:854152 CAPLUS
DOCUMENT NUMBER: 134:231621
TITLE: Serum-dependent effects of tamoxifen and cannabinoids upon C6 glioma cell viability
AUTHOR(S): Jacobsson, S. O. P.; Rongard, E.; Stridh, M.; Tiger, G.; Fowler, C. J.
CORPORATE SOURCE: Department of Pharmacology and Clinical Neuroscience, Umea University, Umea, SE-901 87, Swed.
SOURCE: Biochemical Pharmacology (2000), 60(12), 1807-1813
CODEN: BCPA6; ISSN: 0006-2952
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In the present study, the effects of the combination of tamoxifen ((Z)-2-[p-(1,2-diphenyl-1-butenoxy)-N,N-dimethylamine citrate) and three cannabinoids (A9-tetrahydrocannabinol [A9-THC], cannabidiol, and anandamide [AEA]) upon the viability of C6 rat glioma cells was assessed at different incubation times and using different culturing concns. of fetal bovine serum (FBS). Consistent with previous data for human glioblastoma cells, the tamoxifen sensitivity of the cells was increased as the FBS content of the culture medium was

reduced from 10 to 0.4 and 0%. The cells expressed protein kinase C α and calmodulin (the concentration of which did not change significantly as the FBS concentration was reduced), but did not express estrogen receptors. Δ^9 -THC and cannabidiol, but not AEA, produced a modest reduction in cell viability after 6 days of incubation in serum-free medium, whereas no effects were seen in 10% FBS-containing medium. There was no observed synergy between the effects of tamoxifen and the cannabinoids upon cell viability.

IT 1972-08-3, Δ^9 -Tetrahydrocannabinol

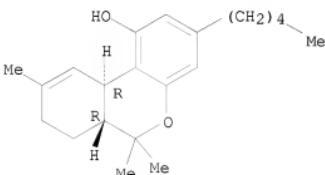
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(serum-dependent effects of tamoxifen and cannabinoids upon C6 glioma cell viability)

RN 1972-08-3 CAPLUS

CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 15 OF 23 USPATFULL on STN

ACCESSION NUMBER: 2007:191333 USPATFULL

TITLE: Cannabinoid derivatives, methods of making, and use thereof

INVENTOR(S): Moore, Bob M. II, Nesbit, MS, UNITED STATES

Ferreira, Antonio M., Memphis, TN, UNITED STATES

Krishnamurthy, Mathangi, Memphis, TN, UNITED STATES

PATENT ASSIGNEE(S): Universty of Tennessee Research Foundation (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2007167514 A1 20070719

APPLICATION INFO.: US 2007-698665 A1 20070126 (11)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2004-850588, filed on 20 May 2004, GRANTED, Pat. No. US 7169942

NUMBER DATE

PRIORITY INFORMATION: US 2003-472316P 20030520 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: O'MELVENY & MYERS LLP, 610 NEWPORT CENTER DRIVE, 17TH FLOOR, NEWPORT BEACH, CA, 92660, US

NUMBER OF CLAIMS: 32

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 13 Drawing Page(s)

LINE COUNT: 2365

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB 1'-substituted cannabinoid derivatives of delta-8-tetrahydrocannabinol, delta-9-tetrahydrocannabinol, and delta-6a-10a-tetrahydrocannabinol that have affinity for the cannabinoid receptor type-1 (CB-1) and/or cannabinoid receptor type-2 (CB-2). Compounds having activity as either agonists or antagonists of the CB-1 and/or CB-2 receptors can be used for treating CB-1 or CB-2 mediated conditions.

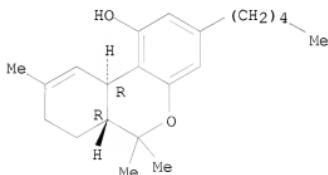
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

IT 1972-08-3, Δ9-Tetrahydrocannabinol
(preparation of cannabinoid derivs. as cannabinoid receptor agonists or antagonists)

RN 1972-08-3 USPATFULL

CN 6H-Dibenzo(b,d)pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L4 ANSWER 16 OF 23 USPATFULL on STN

ACCESSION NUMBER: 2007:147125 USPATFULL

TITLE: Molecular antigen arrays

INVENTOR(S): Bachmann, Martin F., Seuzach, SWITZERLAND

Tissot, Alain, Zurich, SWITZERLAND

Pumpens, Paul, Riga, LATVIA

Cielens, Indulis, Riga, LATVIA

Renhofa, Regina, Riga, LATVIA

PATENT ASSIGNEE(S): Cytos Biotechnology AG, Zurich-Schlieren, SWITZERLAND, CH-8952 (non-U.S. corporation)

NUMBER	KIND	DATE
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PATENT INFORMATION: US 2007128219 A1 20070607

APPLICATION INFO.: US 2006-601687 A1 20061120 (11)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2003-617876, filed on 14 Jul 2003, GRANTED, Pat. No. US 7138252

NUMBER	DATE
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PRIORITY INFORMATION: US 2002-396126P 20020717 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: STERNE, KESSLER, GOLDSTEIN & FOX P.L.L.C., 1100 NEW YORK AVENUE, N.W., WASHINGTON, DC, 20005, US

NUMBER OF CLAIMS: 38

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 11 Drawing Page(s)

LINE COUNT: 5302

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides a composition comprising an AP205 virus

like particle (VLP) and an antigen. The invention also provides a process for producing an antigen or antigenic determinant bound to AP205 VLP. AP205 VLP bound to an antigen is useful in the production of compositions for inducing immune responses that are useful for the prevention or treatment of diseases, disorders or conditions including infectious diseases, allergies, cancer, drug addiction, poisoning and to efficiently induce self-specific immune responses, in particular antibody responses.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

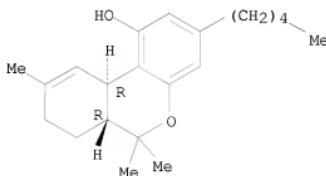
IT 1972-08-3, Tetrahydrocannabinol

(mol. antigen arrays comprising AP205 virus-like particle and antigen for prevention and treatment of cancer, drug addiction, poisoning, infection, and allergy)

RN 1972-08-3 USPATFULL

CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L4 ANSWER 17 OF 23 USPATFULL on STN

ACCESSION NUMBER: 2007:89508 USPATFULL

TITLE:

MODULATION OF NEUROGENESIS BY HDac INHIBITION

INVENTOR(S): Barlow, Carolee, Del Mar, CA, UNITED STATES

Carter, Todd A., San Diego, CA, UNITED STATES

Lorrain, Kym I., San Diego, CA, UNITED STATES

Pires, Jamieson C., San Diego, CA, UNITED STATES

Morse, Andrew, San Diego, CA, UNITED STATES

Gitnick, Dana, San Marcos, CA, UNITED STATES

Treuner, Kai, San Diego, CA, UNITED STATES

Dearie, Alejandro R., Chula Vista, CA, UNITED STATES

PATENT ASSIGNEE(S): BrainCells, Inc., San Diego, CA, UNITED STATES (U.S. corporation)

NUMBER	KIND	DATE
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PATENT INFORMATION: US 2007078083 A1 20070405
APPLICATION INFO.: US 2006-470957 A1 20060907 (11)

NUMBER	DATE
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PRIORITY INFORMATION: US 2005-715219P 20050907 (60)
US 2006-764963P 20060203 (60)
US 2006-785713P 20060324 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834, US

NUMBER OF CLAIMS: 25

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 5466
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The instant disclosure describes methods for treating diseases and conditions of the central and peripheral nervous system by stimulating or increasing neurogenesis. The disclosure includes compositions and methods based on an HDac inhibitory agent alone or in combination with another neurogenic agent to stimulate or activate the formation of new nerve cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 18 OF 23 USPATFULL on STN
ACCESSION NUMBER: 2006227896 USPATFULL
TITLE: Screening assays for cannabinoid-ligand-type modulators
of gpr55
INVENTOR(S): Drmota, Tomas, Molndal, SWEDEN
Greasley, Peter, Molndal, SWEDEN
Groblewski, Thierry, Montreal, CANADA
PATENT ASSIGNEE(S): AstraZeneca AB, Sodertaije, SWEDEN, SE-151 85 (non-U.S.
corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006194260	A1	20060831
APPLICATION INFO.:	US 2004-545851	A1	20040213 (10)
	WO 2004-GB571		20040213
			20050816 PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-448665P	20030218 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	ASTRAZENECA R&D BOSTON, 35 GATEHOUSE DRIVE, WALTHAM, MA, 02451-1215, US	
NUMBER OF CLAIMS:	34	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	9 Drawing Page(s)	
LINE COUNT:	1315	

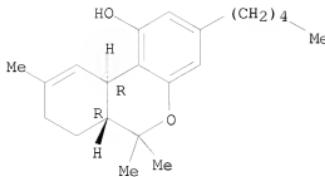
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Screening assays for compounds that modulate the interaction between cannabinoids and the GPR55 receptor are disclosed. Furthermore, a method for determining the selectivity of a test compound against a panel of cannabinoid receptors including GPR55 is disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

IT 1972-08-3, Δ⁹-Tetrahydrocannabinol
(cannabinoid ligand, GPR55 binding to; screening assays for cannabinoid ligand-GPR55 receptor binding modulators)
RN 1972-08-3 USPATFULL
CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-
, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L4 ANSWER 19 OF 23 USPATFULL on STN
 ACCESSION NUMBER: 2004:307917 USPATFULL
 TITLE: Cannabinoid derivatives, methods of making, and use thereof
 INVENTOR(S): Moore, Bob M., II, Nesbit, MS, UNITED STATES
 Ferreira, Antonio M., Memphis, TN, UNITED STATES
 Krishnamurthy, Mathangi, Memphis, TN, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004242593	A1	20041202
	US 7169942	B2	20070130
APPLICATION INFO.:	US 2004-850588	A1	20040520 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-472316P	20030520 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	Edwin V. Merkel, Nixon Peabody LLP, Clinton Square, P.O. Box 31051, Rochester, NY, 14603-1051	

NUMBER OF CLAIMS: 71
 EXEMPLARY CLAIM: 1
 NUMBER OF DRAWINGS: 13 Drawing Page(s)
 LINE COUNT: 2461

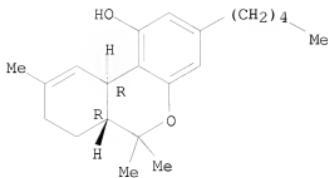
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB 1'-substituted cannabinoid derivatives of delta-8-tetrahydrocannabinol, delta-9-tetrahydrocannabinol, and delta-6a-10a-tetrahydrocannabinol that have affinity for the cannabinoid receptor type-1 (CB-1) and/or cannabinoid receptor type-2 (CB-2). Compounds having activity as either agonists or antagonists of the CB-1 and/or CB-2 receptors can be used for treating CB-1 or CB-2 mediated conditions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

IT 1972-08-3, Δ9-Tetrahydrocannabinol
 (preparation of cannabinoid derivs. as cannabinoid receptor agonists or antagonists)
 RN 1972-08-3 USPATFULL
 CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L4 ANSWER 20 OF 23 USPATFULL on STN

ACCESSION NUMBER: 2004:100750 USPATFULL

TITLE: Molecular antigen arrays

INVENTOR(S): Bachmann, Martin F., Seuzach, SWITZERLAND

Tissot, Alain, Zurich, SWITZERLAND

Pumpens, Paul, Riga, LATVIA

Cielens, Indulis, Riga, LATVIA

Renhofa, Regina, Riga, LATVIA

PATENT INFORMATION: NUMBER KIND DATE

US 2004076611	A1	20040422
US 7138252	B2	20061121

APPLICATION INFO.: US 2003-617876 A1 20030714 (10)

NUMBER DATE

PRIORITY INFORMATION: US 2002-396126P 20020717 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: STERNE, KESSLER, GOLDSTEIN & FOX PLLC, 1100 NEW YORK AVENUE, N.W., WASHINGTON, DC, 20005

NUMBER OF CLAIMS: 51

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 11 Drawing Page(s)

LINE COUNT: 5340

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides a composition comprising an AP205 virus like particle (VLP) and an antigen. The invention also provides a process for producing an antigen or antigenic determinant bound to AP205 VLP. AP205 VLP bound to an antigen is useful in the production of compositions for inducing immune responses that are useful for the prevention or treatment of diseases, disorders or conditions including infectious diseases, allergies, cancer, drug addiction, poisoning and to efficiently induce self-specific immune responses, in particular antibody responses.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

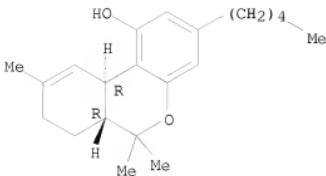
IT 1972-08-3, Tetrahydrocannabinol

(mol. antigen arrays comprising AP205 virus-like particle and antigen for prevention and treatment of cancer, drug addiction, poisoning, infection, and allergy)

RN 1972-08-3 USPATFULL

CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L4 ANSWER 21 OF 23 USPATFULL on STN
 ACCESSION NUMBER: 2004:51617 USPATFULL
 TITLE: Therapy with cannabinoid compounds for the treatment of brain tumors
 INVENTOR(S): Guzman Pastor, Manuel, Madrid, SPAIN
 Sanchez Garcia, Cristina, Madrid, SPAIN
 Galve Roperh, Ismael, Madrid, SPAIN

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004039048	A1	20040226
APPLICATION INFO.:	US 2003-647739	A1	20030825 (10)
RELATED APPLN. INFO.:	Division of Ser. No. US 2001-958960, filed on 27 Nov 2001, ABANDONED A 371 of International Ser. No. WO 2000-ES450, filed on 22 Nov 2000, UNKNOWN		

	NUMBER	DATE
PRIORITY INFORMATION:	ES 2000-323	20000211
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	BAKER & BOTTS, 30 ROCKEFELLER PLAZA, NEW YORK, NY, 10112	
NUMBER OF CLAIMS:	15	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	5 Drawing Page(s)	
LINE COUNT:	469	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The therapy with cannabinoids in the treatment of cerebral tumors involves (intracranial or systematic) administration of (natural or synthetic) cannabinoids to (human or non-human) mammals having cerebral tumors. Activation of the specific receptors of the cannabinoids leads to selective death of the transformed cells. Regression or eradication of the cerebral tumors is achieved without any significant side-effects.

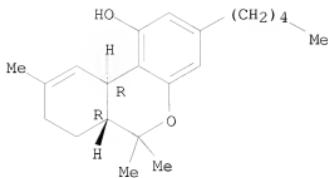
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

IT 1972-08-3, 89-Tetrahydrocannabinol
 (treatment of cerebral tumor using cannabinoids)

RN 1972-08-3 USPATFULL

CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L4 ANSWER 22 OF 23 USPATFULL on STN

ACCESSION NUMBER:

2003:145924 USPATFULL

TITLE:

Packaging of immunostimulatory substances into virus-like particles: method of preparation and use

INVENTOR(S):

Bachmann, Martin, Winterthur, SWITZERLAND

Storni, Tazio, Viganello, SWITZERLAND

Maurer, Patrik, Winterthur, SWITZERLAND

Tissot, Alain, Zurich, SWITZERLAND

Schwarz, Katrin, Schlieren, SWITZERLAND

Meijerink, Edwin, Zurich, SWITZERLAND

Lipowsky, Gerd, Zurich, SWITZERLAND

Pumpens, Paul, Riga, LATVIA

Cielens, Indulis, Riga, LATVIA

Renhofa, Regina, Riga, LATVIA

PATENT ASSIGNEE(S): Cytos Biotechnology AG (non-U.S. corporation)

NUMBER	KIND	DATE
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PATENT INFORMATION:

US 2003099668 A1 20030529

APPLICATION INFO.:

US 2002-244065 A1 20020916 (10)

NUMBER	DATE
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PRIORITY INFORMATION:

US 2001-318994P 20010914 (60)

US 2002-374145P 20020422 (60)

DOCUMENT TYPE:

Utility

FILE SEGMENT:

APPLICATION

LEGAL REPRESENTATIVE:

STERNE, KESSLER, GOLDSTEIN & FOX PLLC, 1100 NEW YORK AVENUE, N.W., SUITE 600, WASHINGTON, DC, 20005-3934

NUMBER OF CLAIMS:

207

EXEMPLARY CLAIM:

1

NUMBER OF DRAWINGS:

60 Drawing Page(s)

LINE COUNT:

7907

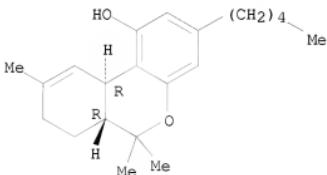
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to the finding that virus like particles (VLPs) can be loaded with immunostimulatory substances, in particular with DNA oligonucleotides containing non-methylated C and G (CpGs). Such CpG-VLPs are dramatically more immunogenic than their CpG-free counterparts and induce enhanced B and T cell responses. The immune response against antigens optionally coupled, fused or attached otherwise to the VLPs is similarly enhanced as the immune response against the VLP itself. In addition, the T cell responses against both the VLPs and antigens are especially directed to the Th1 type. Antigens attached to CpG-loaded VLPs may therefore be ideal vaccines for prophylactic or therapeutic vaccination against allergies, tumors and other self-molecules and chronic viral diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

IT 1972-08-3, Tetrahydrocannabinol
(packaging of immunostimulatory substances and antigens into virus-like
particles for use as vaccines against cancer, autoimmune disease,
allergy and viral infection)
RN 1972-08-3 USPATFULL
CN 6H-Dibenzo[b,d]pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-
, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L4 ANSWER 23 OF 23 USPATFULL on STN
ACCESSION NUMBER: 2003:133508 USPATFULL
TITLE: In vivo activation of antigen presenting cells for
enhancement of immune responses induced by virus like
particles
INVENTOR(S): Bachmann, Martin F., Winterthur, SWITZERLAND
Lechner, Franziska, Zurich, SWITZERLAND
Storni, Tazio, Viganello, SWITZERLAND
PATENT ASSIGNEE(S): Cytos Biotechnology AG (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003091593	A1	20030515
APPLICATION INFO.:	US 2002-243739	A1	20020916 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-318967P	20010914 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	STERNE, KESSLER, GOLDSTEIN & FOX PLLC, 1100 NEW YORK AVENUE, N.W., SUITE 600, WASHINGTON, DC, 20005-3934	
NUMBER OF CLAIMS:	194	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	20 Drawing Page(s)	
LINE COUNT:	6522	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to the finding that stimulation of antigen presenting cell (APC) activation using substances such as anti-CD40 antibodies or DNA oligomers rich in non-methylated C and G (CpGs) can dramatically enhance the specific T cell response obtained after vaccination with recombinant virus like particles (VLPs) coupled, fused or otherwise attached to antigens. While vaccination with recombinant VLPs fused to a cytotoxic T cell (CTL) epitope of lymphocytic choriomeningitis virus induced low levels cytolytic activity only and did not induce efficient anti-viral protection, VLPs injected together with anti-CD40 antibodies or CpGs induced strong CTL activity and full anti-viral protection. Thus, stimulation of APC-activation through antigen presenting cell activators such as anti-CD40 antibodies or CpGs

can exhibit a potent adjuvant effect for vaccination with VLPs coupled, fused or attached otherwise to antigens.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

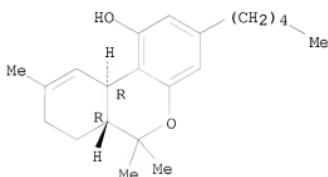
IT 1972-08-3, Tetrahydrocannabinol

(antiviral and antitumor vaccines comprising anti-CD4 antibody or immunostimulatory nucleic acid and antigen-coupled virus-like particles for enhancement of immune responses and activation of antigen-presenting cells)

RN 1972-08-3 USPATFULL

CN 6H-Dibenzo{b,d}pyran-1-ol, 6a,7,8,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-, (6aR,10aR)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



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(FILE 'HOME' ENTERED AT 13:20:44 ON 01 MAY 2008)

FILE 'REGISTRY' ENTERED AT 13:22:01 ON 01 MAY 2008
E "TETRAHYDROCANNABINOL"/CN 25

L1 1 S E3

L2 1 S L1 EXA SAM

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2008

L3 9938 S L2

L4 23 S L3 AND GLIOBLASTOMA

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=> LOG Y

COST IN U.S. DOLLARS

SINCE FILE
ENTRY

FULL ESTIMATED COST

TOTAL
SESSION

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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE
ENTRY

CA SUBSCRIBER PRICE

TOTAL
SESSION

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